Annals of Clinical and Analytical Medicine
Original Research

Comparison of nerve conduction studies and f-waves in the diagnosis of diabetic peripheral neuropathy

Hasan Yaşar[1], Levent Demirtaş[2]
[1] Department of Neurology
[2] Department of Internal Medicine, Faculty of Medicine, Erzincan Binali Yıldırım University, Erzincan, Turkey

Abstract

Aim: The aim of this study was to investigate whether there is a difference in sensitivity in the diagnosis of diabetic peripheral neuropathy and subclinical neuropathy between routine nerve conduction studies and F-waves examined with different numbers of stimuli in patients with diabetes mellitus.

Material and Methods: In the study, eighty patients with diabetes mellitus (forty of them had symptoms of peripheral neuropathy) and forty healthy volunteers as control group were included. Those with peripheral neuropathy symptoms were included in the symptomatic group, and those without peripheral neuropathy symptoms were included in asymptomatic group. All participants underwent routine nerve conduction study.

Results: According to the results of F minimum, F mean and F maximum which were obtained with both 10 consecutive stimuli and 30 consecutive stimuli, there was a statistically significant difference between the symptomatic group and the control group as well as between the asymptomatic group and the control group ($p<0.05$). In F chronodispersion studies, there was no difference between groups at 10 consecutive stimuli and 30 consecutive stimuli ($p > 0.05$).

Discussion: F minimum, F mean, F maximum tests were more sensitive than routine nerve conduction study in detecting asymptomatic diabetic peripheral neuropathies. The results of the tests with 10 and 30 stimuli were similar. F chronodispersion was found to be insensitive in the diagnosis of diabetic peripheral neuropathy and subclinical neuropathies.

Keywords

Diabetic Polyneuropathy, Electrophysiology, Electrical Nerve Stimulation, Nerve Conduction, F-waves

DOI: 10.4328/ACAM.21495 Received: 2022-11-13 Accepted: 2022-12-24 Published Online: 2023-01-05 Printed: 2023-02-01 Ann Clin Anal Med 2023;14(2):152-156
Corresponding Author: Hasan Yaşar, Department of Neurology, Faculty of Medicine, Erzincan Binali Yıldırım University, 24100, Erzincan, Turkey.
E-mail: yasar_hsan@hotmail.com P: +90 446 226 18 18 / +90 505 631 57 19 F: +90 446 226 18 19
Corresponding Author ORCID ID: https://orcid.org/0000-0001-8601-1277
This study was approved by the Ethics Committee of Erzincan Binali Yildirim (Date: 2021-12-06, No: 13/06)

Introduction

Diabetes mellitus (DM) is one of the most important health problems recently[1]. It is a cause of mortality and morbidity due to complications in many organs and systems. Among the most important complications are neuropathy, nephropathy and retinopathy. Diabetic Peripheral Neuropathy (DPN) is one of the most common complications[2]. DM affects nerves both acutely and chronically. Two theories have been proposed regarding the formation of neuropathy. Polyol pathway activation increases due to hyperglycemia and leads to neuropathy by causing sorbitol accumulation. Another hypothesis is endoneurial hypoxia due to vascular causes[1]. Both myelinated and unmyelinated nerves are affected. Affecting the myelinated fibers causes numbness in the distal extremities, unbalanced gait, muscle atrophy and weakness. Whereas painful neuropathy and autonomic symptoms occur when unmyelinated fibers are affected[2]. If DPN is not diagnosed and treated early, it causes foot ulceration and amputation, leading to disability[3]. DPN is seen as symmetrical sensorimotor polyneuropathy[1]. It occurs as both axonal and demyelinating neuropathy[4]. Sensory symptoms begin in the toes and affect the upper extremities over time. Motor symptoms appear later than sensory symptoms. After the sensory nerves are affected, patients experience a decrease in pain sensation, sensation of pins and needles, tingling, pain in situations where pain should not normally occur (allodynia), excessive pain (hyperalgesia), hot-cold discrimination disorder[1].

In the presence of clinical findings, electrophysiological examinations and nerve conduction studies are performed to detect DPN[5]. In DM patients, the presence of neuropathy can be revealed subclinically in nerve conduction studies without clinical findings[6]. There is controversy regarding the fact that routine sensory and motor nerve conduction examinations are insufficient to detect DPN and subclinical neuropathies, and that F-wave examinations can reveal abnormalities before routine conduction examinations and are more sensitive[7].

The F-wave is one of the most fundamental studies of clinical neurophysiology[8]. It is called the F-wave because it was first shown on the lower extremities[6]. It is produced by anterior horn cells. It demonstrates excitability of spinal motor neurons. It is highly sensitive in detecting pathology of proximal segments[6]. The amount of stimulus that should be administered supramaximally is controversial. In some studies, 10, 16, 20, 32 stimuli were applied and it was suggested that increasing the amount of stimulus gave more accurate results. However, some studies suggest that increasing the amount of stimulus is not an important factor[8].

The aim of this study was to examine both F-waves at different stimulus numbers (10,30) between diabetic patients with and without DPN symptoms and control groups and to examine and compare whether there is a difference in sensitivity between routine nerve conduction examinations and F-wave examinations.

Material and Methods

It was ensured that the skin temperature is 32-36 degrees[9]. The examination was done by the same person who was blind to clinical information of the patients. Informed consent form prepared in accordance with the Declaration of Helsinki was obtained from all patient and control groups and the approval of the local ethics committee was received, (Date:06.12.2021, Number:13/06). Patients with renal failure, substance abuse, a history of cerebrovascular, cardiovascular disease, those with cardiac pacemaker, neurotoxic drug use, drug use affecting the nervous system, history of malignancy, thyroid disease, cervical and lumbar trauma and disc disease were excluded from the study[5, 10, 11].

Symptoms of peripheral neuropathy include unpleasant abnormal sensations in the distal extremities, touch, pain, decreased sensation of temperature, burning sensation in the feet, tingling sensation, pins and needles, electrification, cramp-like sensation, decreased or absent ankle reflex, muscle weakness. Michigan Neuropathy Screening Instrument was used to differentiate between symptomatic and asymptomatic patients. According to the scale, those with a clinical examination score >2 were considered symptomatic, and those ≤2 were considered asymptomatic[12]. A total of 120 people, including 40 DM patients with peripheral neuropathy symptoms, 40 DM patients without peripheral neuropathy symptoms, and 40 healthy controls, were included in the study. The examinations were made in the lower extremities.

Nerve conduction study and F-wave examinations were made with the Medelec Synergy model device. Sensory nerve conduction study was performed antidromically. Peak amplitude values and conduction velocities were measured in sensory nerve examinations and were compared between the patients and control groups. Onset latency is the time from the stimulus to the first negative deviation of the baseline for a biphasic sensory nerve action potential (SNAP) or the first positive peak for a triphasic SNAP. The amplitude was evaluated as the amplitude measured between the first electronegative peak and the second electropositive peak. In motor nerve examinations, compound muscle action potentials (CMAP) recorded with distal and proximal stimulation, motor distal latency, peak amplitude values, and conduction velocities were measured and compared between groups. Latency was evaluated as the time elapsed between the stimulus and the point where the potential left the baseline in the electronegative direction. The amplitude was evaluated as the amplitude of the oscillation between the baseline and the electronegative peak. When calculating the motor nerve conduction velocity, the differential latency was obtained by subtracting the distal latency of the CMAP obtained with distal stimulation from the proximal latency of the CMAP obtained with proximal stimulation, and the distance between the two stimulation points was divided by this differential latency and the conduction velocity was calculated[13].

For sural nerve conduction examination, the recording electrode was placed behind the lateral malleolus. Stimulation was performed from 12 cm proximal to the recording electrode, from the middle posterior part of the leg[10]. The recording electrode was placed on the extensor digitorum brevis muscle for peroneal motor nerve conduction examination. The peroneal nerve was stimulated distally at the ankle level, laterally from the tibialis anterior muscle tendon, and proximally from the distal of the fibula head. For tibial nerve motor conduction examination, the recording electrode was placed on the abductor hallucis muscle.

The tibial nerve was stimulated from behind the medial mallous and from the popliteal fossa [14].

For the F-wave study, the electrodes were placed as for the motor nerve conduction study. For tibial and peroneal F-wave examination, supramaximal stimulus was applied from the retro-maleolar region and lateral to tibialis anterior tendon at ankle, respectively[10]. For F-wave recording, 10 (F-10) and 30 (F-30) supramaximal stimuli were obtained separately by antidromic application. F-waves were recorded while the patients were at rest. As a result of supramaximal stimulation, it was distinguished by its longer, variable latency, low shape and amplitude after the M wave. Regardless of whether the initial deflection is positive or negative, the initial latency was accepted as the starting point of the response. Minimum latency (F min), maximum latency (F max), mean latency (F mean) and chronodispersion (F cd) measurements of the F-wave were made. Waves greater than 20 μV were considered as F- waves. F-wave measured as peak-to-peak. The F-wave with the shortest latency was accepted as F min. The F mean was obtained by dividing the sum of the initial latencies by the number of F-waves[10, 11]. F max was accepted as the longest duration F-wave. F cd was accepted as the difference between the maximum and minimum F-wave. The latency of the F-wave is directly proportional to the limb length. Height can also be used instead of limb length[15]. In this study, height was used in the evaluation between groups. F-wave obtained with distal stimulus from the ankle. Signals were recorded at an amplification of 500μV/div during a 100-ms period following the stimulus[16].

Weight and height measured in the lightest clothing possible and without shoes. The study was carried out in Type-2 DM patients.

Biochemical analysis

Venous blood samples for biochemical analysis were obtained after at least 10 hours of fasting and before any drug administration. All biochemical analyzes were performed in Local Central Biochemistry Laboratory.

Statistical analysis

Statistical analysis was performed using the IBM SPSS v. 19 package program (IBM Corp., Armonk, USA). The statistical level of significance for all tests was considered to be 0.05. The results were presented for continuous variables as mean ± standard deviation (sd), median and minimum– maximum. The normality of distribution for continuous variables was confirmed with the Shapiro-Wilk test. For comparison of independent continuous variables between 3 groups, analysis of variance (ANOVA) or Kruskal-Wallis test was used. Homogeneity of variances was confirmed with Levene's test. While comparing 2 groups, Tukey HSD, Games-Howell or Mann-Whitney U test was used as post-hoc test. Pearson's chi-square test was used for the analysis of qualitative groups. Bonferroni adjustment was made for nonparametric pairwise multiple comparison tests in independent groups.

Ethical Approval

Ethics Committee approval for the study was obtained.

Results

Descriptive statistics values between symptomatic, asymptomatic and control groups are shown in Table 1. No significant difference was observed between the groups in terms of gender, age, weight, height, body mass index (BMI), duration of diabetes and HbA1c levels (p>0.05).

In the examination of the sural sensory, the common peroneal motor and the tibial motor nerves, there was a significant difference between symptomatic DM patients and the control

Table 1. Descriptive statistics.

Parameter	DM (S)	DM (AS)	Control	p-value
Gender	Male:17 (42,5%)	Male:23 (57,5%)	Male:17 (42,5%)	>0,05[x2]
	Female:23 (57,5%)	Female:17 (42,5%)	Femal:23 (57,5%)	
Age (year)	59,57±8,19	55,65±10,67	55,07±8,21	>0,05[a]
	59,00(43-79)	57,00(30-75)	56,00(35-75)	
Weight (kg)	85,33±14,00	81,80±13,54	78,93±10,47	>0,05[a]
	85,00(59-120)	81,00(56-120)	78,00(57-100)	
Height (m)	1,67±0,08	1,66±0,07	1,68±0,07	>0,05[a]
	1,67(1,53-1,86)	1,67(1,54-1,82)	1,67(1,55-1,85)	
BMI(kg/m2)	30,54±5,03	29,47±4,32	28,02±3,96	>0,05[b]
	30,04(21,0-44,1)	29,41(21,2-41,5)	27,33(21,9-39,1)	
Duration of diabetes (year)	12,22±8,06	8,70±4,26		>0,05[c]
	10,00(2-33)	8,50(1-20)		
HbA1c (%)	9,14±2,25	8,41±2,26		>0,05[c]
	9,00(6,30-15,70)	7,60(6,20-16,90)		

a: One-way anova test, b:Kruskall-Wallis test. c:Mann-Whitney U test, x^2: Pearson's chi-square test. BMI: Body mass index, DM: Diabetes mellitus, (S): Symptomatic, (AS): Asymptomatic. HbA1c: Glycosylated hemoglobin.

Table 2. Sensory and motor nerve conduction values of groups

Variable		DM (S)	DM (AS)	Control (C)	p-value	
					S vs C	AS vs C
SN	SNAP (μV)	10,11±4,38	13,72±6,97	17,25±8,80	0,000	0,205[b]
		10,15 (0,80-22,90)	12,10 (2,10-36,70)	15,35 (3,00-41,20)		
	Velocity (m/s)	47,99±7,34	55,46±9,22	58,46±7,21	0,000	0,218[a]
		48,15 (31,10-63,30)	56,35 (31,50-69,00)	57,80 (44,10-74,10)		
	Latence (ms)	2,53±0,62	2,16±0,61	2,09±1,28	0,000	0,266[b]
		2,50 (1,50-4,20)	2,05 (1,20-3,80)	1,90 (1,40-9,70)		
CPMN	CMAP (mV)	3,96±1,75	4,21±1,23	5,23±2,07	0,021	0,271[b]
		3,90 (1,10-8,10)	4,22 (2,00-7,00)	4,65 (2,20-9,80)		
	Velocity (m/s)	44,59±6,77	50,48±5,97	50,89±4,92	0,000	0,939[a]
		43,45 (32,20-58,00)	51,00 (39,40-66,00)	51,40 (41,50-66,70)		
	Latence (ms)	4,04±1,06	3,60±0,61	3,45±0,55	0,008	0,895[b]
		3,85 (2,60-8,55)	3,65 (2,80-5,50)	3,30 (2,40-4,60)		
TMN	CMAP (mV)	4,71±2,20	6,66±2,28	8,28±3,66	0,000	0,053[a]
		4,25 (1,40-9,80)	6,45 (1,90-11,10)	7,45 (2,60-18,10)		
	Velocity (m/s)	42,14±7,17	45,95±5,38	47,62±4,58	0,001	0,796[b]
		41,65 (29,00-55,00)	46,50 (34,40-57,60)	47,30 (40,20-65,20)		
	Latence (ms)	5,29±1,42	4,65±1,00	4,27±0,80	0,001	0,300[b]
		5,05 (3,10-9,20)	4,50 (2,90-7,70)	4,15 (2,80-6,50)		

a:One-way Anova test, b:Kruskall-Wallis test, mean±sd, median (minimum-maximum), DM: Diabetes mellitus, (S): Symptomatic, (AS): Asymptomatic, SN:Sural nerve, CPMN:Common peroneal motor nerve, TMN:Tibial motor nerve, SNAP: Sensory nerve action potential, CMAP:Compound muscle action potential.

Table 3. F-10, F-30 stimulation

Variable	DM (S)	DM (AS)	Control (C)	p-value	
				S vs C	AS vs C
CPFmin (ms)	49,39±6,34	48,19±5,64	44,05±3,87	0,000[1]	0,001[a]
	48,00 (40,20-64,85)	46,95 (39,70-63,00)	44,30 (37,20-52,10)		
	47,58±6,96	45,63±5,07	42,26±3,63	0,000[2]	0,014[b]
	46,80 (31,35-66,95)	44,80 (38,35-62,00)	42,60 (34,80-49,60)		
CPFmax (ms)	63,86±7,16	63,81±6,53	60,18±5,98	0,029[1]	0,017[b]
	62,43 (49,34-80,25)	64,10 (51,10-81,00)	59,15 (50,00-82,30)		
	65,39±6,85	65,50±7,19	60,73±6,11	0,009[2]	0,011[b]
	62,90 (54,70-84,50)	65,70 (55,10-84,00)	60,60 (47,00-80,50)		
CPFmean (ms)	52,86±6,34	50,07±5,85	46,93±4,55	0,000[1]	0,037[a]
	52,70 (42,40-68,82)	49,50 (40,80-66,60)	46,40 (37,40-56,70)		
	52,73±7,16	51,54±5,98	46,73±4,81	0,000[2]	0,002[a]
	52,73 (39,50-68,06)	51,95 (41,90-66,40)	47,10 (38,20-55,50)		
CPFcd (ms)	14,47±5,22	16,21±4,41	16,12±4,15	>0,05[1]	>0,05[b]
	15,20 (2,75-27,50)	15,70 (7,00-27,30)	15,75 (9,90-30,20)		
	17,80±5,50	18,92±5,53	18,46±4,76	>0,05[2]	>0,05[a]
	18,06 (6,35-31,20)	18,00 (9,90-31,50)	18,40 (8,20-30,90)		
TFmin (ms)	51,82±7,47	50,14±6,03	45,60±4,45	0,000[1]	0,002[b]
	50,35 (39,40-69,40)	49,55 (40,30-64,20)	44,60 (39,00-61,40)		
	50,34±7,21	47,52±5,46	43,96±4,12	0,000[2]	0,011[b]
	49,20 (35,05-69,10)	47,15 (37,20-60,00)	42,60 (37,90-52,90)		
TFmax (ms)	68,21±9,58	67,15±8,60	62,95±7,51	0,010[1]	0,045[b]
	68,40 (53,65-96,80)	66,40 (51,15-89,00)	61,50 (50,70-86,00)		
	69,43±9,70	68,97±8,52	64,09±7,43	0,016[2]	0,032[b]
	69,17 (53,65-97,80)	67,90 (54,10-94,00)	61,90 (49,30-78,90)		
TFmean (ms)	56,58±7,88	54,41±6,19	49,43±4,02	0,000[1]	0,001[b]
	55,70 (44,57-78,50)	53,55 (44,00-67,00)	47,90 (43,20-57,70)		
	56,08±7,88	52,81±6,08	48,81±5,03	0,000[2]	0,017[a]
	54,70 (43,73-77,90)	52,40 (42,80-68,40)	47,40 (39,30-61,00)		
TFcd (ms)	16,39±6,53	17,30±6,14	17,35±6,15	>0,05[1]	>0,05[a]
	17,35 (4,40-30,10)	17,10 (1,55-35,40)	17,00 (7,20-36,90)		
	19,09±5,93	20,86±7,29	20,13±6,14	>0,05[2]	>0,05[a]
	19,20 (4,85-32,40)	19,75 (3,20-37,00)	21,00 (7,50-33,80)		

[1]F-10,[2]F-30 Stimulation. a: One-way Anova test, b:Kruskall-Wallis test, mean±sd, median (minimum-maximum), CPFmin: Common peroneal nerve F minimum, CPFmax: Common peroneal nerve F maximum, CPF mean: Common peroneal nerve F mean, CPFcd: Common peroneal nerve F chronodispersion, TFmin: Tibial nerve F minimum, TFmax: Tibial nerve F maximum, TFmean: Tibial nerve F mean, TFcd: Tibial nerve F chronodispersion, (S): Symptomatic, (AS): Asymptomatic.

group in terms of amplitude, conduction velocity and latency values (p<0.05); however, there was no significant difference between asymptomatic patients and control group (p>0.05), (Table 2).

As shown in Table 3, groups were examined separately with 10 and 30 stimulus numbers. When the common peroneal motor nerve and the tibial motor nerve were examined in terms of F min, F max, and F mean in both 10 and 30 stimulus numbers, it was found that there was a significant difference between the symptomatic group and the control group, and between the asymptomatic group and the control group (p<0.05). There was no significant difference between the groups in the examination of F cd values (p>0.05).

Discussion

DPN occurs due to metabolic and microvascular causes such as hyperglycemia and decreased Na/K ATPase enzyme activity, increased anaerobic glycolysis, oxidative stress, mitochondrial dysfunction, polyol pathway activation, and microangiopathy[7]. The majority of patients develop DPN despite good glucose control. However, the first choice in the treatment of DPN is glycemic control[1, 17].

Motor and sensory nerve conduction examinations are performed for the diagnosis of DPN[1]. Studies have shown that there is increased sensitivity in the proximal segments of the nerves in the early stages of diabetes and it is necessary to examine the proximal segments for the diagnosis of subclinical diabetic neuropathy[11]. It has been suggested that sensory and motor examinations are insufficient to examine the long proximal segment of the nerve, therefore routine nerve conduction studies are insufficient for the early diagnosis of diabetic subclinical neuropathy[18].

When the motor nerve is stimulated from any point, it creates a compound muscle action potential in the muscle tissue with orthodromic conduction. The antidromic conduction stimulates some cells of the anterior horn. The impulses from the anterior horn cells cause an additional small depolarization of the muscle. This depolarization is called F-waves[6]. The F-wave is the late wave that occurs after supramaximal stimulation of the motor nerve. It also gives information about the state of motor neurons in the cervical and lumbosacral cords. The afferent and efferent pathway of the F-wave is the alpha motor neuron[3]. Ensuring glycemic control has the fastest effect on F-waves and is the most important follow-up investigation that gives the fastest response to glycemic changes[7].

DPN begins earlier in the lower extremities than in the upper extremities. It has been suggested that sural sensory potential is the earliest examination to show abnormality in diabetes. It shows abnormality before the peroneal and tibial motor nerves. However, it has been suggested that the F-wave is affected before the sural nerve[19]. F min reflects the fastest motor neurons[20]. F min elongation is explained by loss of the most rapid axons or decreased excitability of anterior horn cells[4]. F mean is an average measure of all axons[15]. F max reflects slow conducting neurons of motor neurons[21]. A few of the anterior horn cells are stimulated with each stimulation. For this reason, there are debates about whether at least 10 or 20 stimulations will give accurate results. In many studies, it was seen that between 10 and 32 stimuli were used, and in a few studies, stimuli were given below or above this number of stimuli[4, 6]. While 5 or 10 measurements are sufficient for the objective criterion for F mean, it has been reported that more stimuli should be applied for F min, F max and F cd. It is recommended that the number of stimuli applied should be at least 30 in order to evaluate F cd correctly[6].

There are studies reporting that the most sensitive test for the

early diagnosis of diabetic subclinical neuropathy is the F min examination, and the lower extremity tibial and peroneal F min examination is the most sensitive test[1]. In some studies, F min and F mean examinations in the lower extremities were shown to be the most sensitive findings[11]. In one study, F max, and F mean values were found to be more sensitive than F min values when the healthy group and diabetic patients were compared, but this study was reported as a small study[22].

It has been shown that F-waves are associated with the duration of diabetes and HbA1c levels and are affected by these parameters. It has been shown that F-waves are significantly prolonged in patients with neurological examination findings and sensory symptoms[6].

In present study, 10 and 30 stimuli were applied separately to the patient and control groups. The patient and control groups were compared with different numbers of stimuli. In the study, there was no significant difference in terms of F cd values in 10 and 30 stimuli. Thus, F cd was not sensitive to detect symptomatic or non-symptomatic patients with diabetes and does not support studies suggesting that F cd is sensitive. In routine nerve conduction examination, symptomatic patients were significantly different from the control group, but asymptomatic patients and control group were similar. Therefore, routine nerve conduction examinations were insufficient for the early detection of asymptomatic patients. There were significant differences between symptomatic patients and control groups and between asymptomatic patients and control groups in both 10 and 30 stimuli; F-wave examinations were more sensitive than routine examination in early detection of asymptomatic patients and subclinical neuropathies. Examinations at different stimulus numbers did not make any difference in sensitivity. Sensitivity did not change in F min, F max and F mean examinations at 10 and 30 stimuli.

Conclusion

Routine nerve conduction examination detected symptomatic patients, but it was insufficient to detect asymptomatic patients. F-wave examination was more sensitive than routine examination in detecting subclinical neuropathy in asymptomatic patients. F min, F max, F mean did not show any difference in sensitivity at different number of stimuli. F cd examination was similar in diabetes and control groups and its use in detection of peripheral neuropathies was not sensitive.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding: *None*

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References

1. Juster-Switlyk K ,Smith AG. Updates in diabetic peripheral neuropathy, F1000Res. 2016; 5.

2. Weng YC, Tsai SS, Lyu RK, Chu CC, Ro LS, Liao MF, et al. Diabetic distal symmetrical polyneuropathy: Correlation of clinical, laboratory, and electrophysiologic studies in patients with type 2 diabetes mellitus, J Diabetes Res. 2020; 2020:1-11.

3. Sathya GR, Krishnamurthy N, Veliath S, Arulneyam J ,Venkatachalam J. F wave index: A diagnostic tool for peripheral neuropathy, Indian J Med Res. 2017; 145(3):353-7.

4. Wang ZL, Cui L, Liu M, Zhang K, Liu S, Ding Q, et al. Reassessment of split-leg signs in amyotrophic lateral sclerosis: Differential involvement of the extensor digitorum brevis and abductor hallucis muscles, Front Neurol. 2019; 10:565.

5. Trujillo-Hernández B, Huerta M, Trujillo X, Vásquez C, Pérez-Vargas D ,Millán-Guerrero RO. F-wave and h-reflex alterations in recently diagnosed diabetic patients, J Clin Neurosci. 2005; 12(7):763-6.

6. De Souza RJ, De Souza A ,Nagvekar MD. F-wave studies in diabetes mellitus: A comparison between pre-symptomatic and symptomatic patients with distal sensory polyneuropathy, Neurology, Psychiatry and Brain Research. 2018; 30:125-31.

7. Kikkawa Y, Kuwabara S, Misawa S, Tamura N, Kitano Y, Ogawara K, et al. The acute effects of glycemic control on nerve conduction in human diabetics, Clin Neurophysiol. 2005; 116(2):270-4.

8. Fisher MA. Comparison of automated and manual f-wave latency measurements, Clin Neurophysiol. 2005; 116(2):264-9.

9. Tankisi H, Pugdahl K, Beniczky S, Andersen H ,Fuglsang-Frederiksen A. Evidence-based recommendations for examination and diagnostic strategies of polyneuropathy electrodiagnosis, Clin Neurophysiol Pract. 2019; 4:214-22.

10. Pinheiro DS, Manzano GM ,Nóbrega JA. Reproducibility in nerve conduction studies and f-wave analysis, Clin Neurophysiol. 2008; 119(9):2070-3.

11. Karsidag S, Morali S, Sargin M, Salman S, Karsidag K ,Us O. The electrophysiological findings of subclinical neuropathy in patients with recently diagnosed type 1 diabetes mellitus, Diabetes Res Clin Pract. 2005; 67(3):211-9.

12. Kaymaz S, Alkan H, Karasu U ,Çobankara V. Turkish version of the michigan neuropathy screening instrument in the assessment of diabetic peripheral neuropathy: A validity and reliability study, Diabetol Int. 2020; 11(3):283-92.

13. Palve SS ,Palve SB. Impact of aging on nerve conduction velocities and late responses in healthy individuals, J Neurosci Rural Pract. 2018; 9(1):112-6.

14. Pan H, Lin J, Chen N, Jian F, Zhang Z, Ding Z, et al. Normative data of f-wave measures in china, Clin Neurophysiol. 2013; 124(1):183-9.

15. Nobrega JA, Pinheiro DS, Manzano GM ,Kimura J. Various aspects of f-wave values in a healthy population, Clin Neurophysiol. 2004; 115(10):2336-42.

16. Jerath NU, Aul E, Reddy CG, Azadeh H, Swenson A ,Kimura J. Prolongation of f-wave minimal latency: A sensitive predictor of polyneuropathy, Int J Neurosci. 2016; 126(6):520-5.

17. Maekawa T, Tadaki H, Sasase T, Motohashi Y, Miyajima K, Ohta T, et al. Pathophysiological profiles of sdt fatty rats, a potential new diabetic peripheral neuropathy model, J Pharmacol Toxicol Methods. 2017; 88(Pt 2):160-6.

18. Bae JS ,Kim BJ. Subclinical diabetic neuropathy with normal conventional electrophysiological study, J Neurol. 2007; 254(1):53-9.

19. Sekiguchi K, Kohara N, Baba M, Komori T, Naito Y, Imai T, et al. Aldose reductase inhibitor ranirestat significantly improves nerve conduction velocity in diabetic polyneuropathy: A randomized double-blind placebo-controlled study in japan, J Diabetes Investig. 2019; 10(2):466-74.

20. Puksa L, Eeg-Olofsson KE, Stålberg E ,Falck B. Reference values for f wave parameters in healthy 3-20 year old subjects, Clin Neurophysiol. 2011; 122(1):199-204.

21. Puksa L, Stålberg E ,Falck B. Reference values of f wave parameters in healthy subjects, Clin Neurophysiol. 2003; 114(6):1079-90.

22. Pan H, Jian F, Lin J, Chen N, Zhang C, Zhang Z, et al. F-wave latencies in patients with diabetes mellitus, Muscle Nerve. 2014; 49(6):804-8.

How to cite this article:

Hasan Yaşar, Levent Demirtaş*. Comparison of nerve conduction studies and f-waves in the diagnosis of diabetic peripheral neuropathy. Ann Clin Anal Med 2023;14(2):152-156*

This study was approved by the Ethics Committee of Erzincan Binali Yildirim (Date: 2021-12-06, No: 13/06)